www.nature.com/jea

Environmental tobacco smoke exposure in children: parental perception of smokiness at home and other factors associated with urinary cotinine in preschool children

DOLORES JURADO,^a CARMEN MUÑOZ,^a JUAN DE DIOS LUNA^b AND MILAGROS FERNÁNDEZ-CREHUET^a

^aDepartment of Preventive Medicine and Public Health, University of Granada, Spain ^bDepartment of Statistics, University of Granada, Spain

Parental smoking behavior at home and sociodemographic variables may influence exposure to environmental tobacco smoke (ETS) in children. A sample of 115 preschool children aged 3–6 years was enrolled in this study. ETS exposure was evaluated through a questionnaire about parents' smoking behavior and determinations of urinary cotinine — a biomarker of exposure — in children. Bivariate and multiple regression analyses were used to evaluate the association between the smoking behavior of each parent at home, sociodemographic factors and cotinine levels in children. The parental perception of smokiness in the home was significantly associated with urinary cotinine in children (r-partial coefficient = 0.324; P < 0.002). The father's education, mother's smoking status, and day of the week when urine was sampled (Tuesday) were also independently associated with levels of cotinine. These four variables explained 26.4% of the variance in the cotinine levels of children. In designing educational programs to reduce passive smoking among children, it is necessary to take into account those factors related with cotinine levels in children. The parents (smokers and nonsmokers) about the smokiness in the home could also be a useful indicator of the cotinine in children exposed to environmental tobacco smoke in the household.

Journal of Exposure Analysis and Environmental Epidemiology (2004) 14, 330-336. doi:10.1038/sj.jea.7500329

Keywords: environmental tobacco smoke, passive smoking, cotinine, child, preschool, smoking, parent-child relations.

Introduction

Exposure to environmental tobacco smoke (ETS) is an important preventable cause of morbidity and mortality in children (Aligne and Stoddard, 1997). Health effects of children's passive smoking, through prenatal and/or postnatal exposure, have been well documented (US Environ-Protection Agency, 1992; World mental Health Organization, 1999). Parental smoking at home is the single most important source of passive exposure in childhood (Cook et al., 1994; Dell'Orco et al., 1995; Bakoula et al., 1997; Irvine et al., 1997; Winkelstein et al., 1997; Jordaan et al., 1999), although other family members, caregivers, visitors or friends may also contribute to the level of smokiness in the home (Cook et al., 1994; Irvine et al., 1997; Preston et al., 1997; Ownby et al., 2000). The combination of a questionnaire about parents' smoking behavior and determinations of cotinine (a biomarker of exposure) in organic fluids of children can be applied in epidemiologic studies to assess ETS exposure among children (Dell'Orco et al., 1995; Bakoula et al., 1997; Irvine et al., 1997; Peterson et al., 1997; Preston et al., 1997; Winkelstein et al., 1997; Bahçeciler et al., 1999; Jordaan et al., 1999; Oddoze et al., 1999; Seifert et al., 2002).

The cotinine levels in children, measured in saliva or urine, increase with the number of smoking parents (Cook et al., 1994; Irvine et al., 1997; Seifert et al., 2002), the number of smokers in the household (Jordaan et al. 1999), the number of cigarettes/day that the parents smoke at home (Bakoula et al., 1997; Irvine et al., 1997; Oddoze et al., 1999) and the number of cigarettes/day that household members (parents and others) smoke at home (Preston et al., 1997; Winkelstein et al., 1997; Manino et al., 2001; Callais et al., 2003). Moreover, the cotinine is substantially reduced when parents do not smoke in the presence of their children (Bakoula et al., 1997; Irvine et al., 1997; Seifert et al., 2002), and increases progressively with the frequency of smoking in their presence (Irvine et al., 1997).

Urinary cotinine has been found to be inversely related to the age of the child (Bakoula et al., 1997; Irvine et al., 1997; Preston et al., 1997) and to be greater in girls than in boys (Jarvis et al., 1992; Cook et al., 1994; Bakoula et al., 1997; Jordaan et al., 1999). Other studies, however, have not observed the effect of age (Cook et al., 1994; Dell'Orco et al.,

Address all correspondence to: D. Jurado, Departamento de Medicina Preventiva y Salud Pública, Facultad de Medicina, Avda. Madrid, 11, 18012 Granada, Spain. Tel: +34-958-243544. Fax: +34-958-246118. E-mail: djurado@.ugr.es

Received March 2003; accepted 28 October 2003

1995) or gender (Dell'Orco et al., 1995; Irvine et al., 1997). In addition, urinary cotinine in children has been associated with socioeconomic factors such as the level of education of the parents (Dell'Orco et al., 1995; Irvine et al., 1997; Jordaan et al., 1999; Manino et al., 2001), social class (Jarvis et al., 1992; Cook et al., 1994; Irvine et al., 1997), crowding index (Jarvis et al., 1992; Dell'Orco et al., 1995; Irvine et al., 1997), number of persons per household (Jordaan et al., 1999), number of rooms in the home (Dell'Orco et al., 1995; Manino et al., 2001) and total floor surface area (Bakoula et al., 1997).

Among reducing passive smoking in children, the health education of parents is fundamental. For this reason, it is necessary to assess parental smoking behavior in the home as well as sociodemographic factors that may influence exposure. The objective of this study was to evaluate exposure to ETS in the household in a sample of preschool children aged 3–6 years through a questionnaire about parents' smoking behavior at home and determinations of urinary cotinine levels in the children; then to analyze the association between the smoking behavior of each parent, sociodemographic factors and urinary cotinine in children. This is the primary aim of a more extensive study in which we also analyze the effects of ETS on the respiratory health of children 3–6 years of age.

Methods

Study Population

The study was carried out in the city of Granada, Spain, during April and May of 1999. From a complete list of all the primary schools offering preschool education in Granada, a cluster sample was taken in two stages: first of the schools, then of the children (6% precision, confidence interval 95%). The resulting population was a total of 521 children aged 3-6 years, randomly chosen from 25 different preschools. The present study involved 166 children, randomly chosen from the total sample of 521. A note was sent to the parents requesting their informed consent and participation. Of the 166 children, 115 participated (69.3%)in both the questionnaire and the cotinine determinations. The possible differences between the sample of children participating in the present study and those that did not, with regard to a group of variables of interest (age, gender, paternal smoking, maternal smoking and number of smoking parents) were analyzed. There were no significant differences among groups, the final sample being representative of the total sample.

Questionnaire

Data were obtained from the children's parents using a selfreporting questionnaire about smoking behavior. The questionnaires were distributed to the parents by the children's teachers. The smoking status of each parent was classified as: never smokers, ex-smokers, current smokers, or occasional smokers (U.S. Department of Health and Human Services, Center for Disease Control, 1994). For each child, the number of parents who were current or occasional smokers was evaluated (none, one, both). Current smoking fathers and mothers were asked about the following practices at home: smoking in the home in the (main) living room in the presence of offspring (yes/no), smoking in another part of the house such as the patio or balcony (yes/no), and number of cigarettes/day smoked by the father or mother or the family (parents and other usual residents) inside the house (0, 1-5, 6-10, 11-20, 21-30). In addition, the perception of all the parents (smokers and nonsmokers) as to the smokiness of the home was evaluated with this query: "How would you classify the atmosphere of tobacco smoke usually present in your home (none at all, a little, quite a bit, a lot)?".

Besides the above variables, sociodemographic data were analyzed: age and gender of the children; parents' educational level; and crowding index, which was calculated by dividing the number of people in the house by the number of rooms (not counting kitchen and bathrooms). In keeping with Jarvis et al. (1992), three levels of crowding were established: low, less than one person per room; medium, one person; and high, more than one person.

Determination of Urinary Cotinine in Children

One urine sample was collected from each child between Tuesday and Friday; no samples were gathered on Mondays because of organizational difficulties for the schools. The samples were stored at -20° C and were sent to the laboratory for radioimmunoassay (Henderson et al., 1989). Calibration was set at 6 points, and concentrations ranged between 0 and 15.000 ng/ml of cotinine. Results were expressed as cotinine/creatinine ratios (CCR) in ng/mg. Assays were repeated when initial values were over 100 ng/ mg creatinine; levels of cotinine were reported as negative when values were 0 ng/mg creatinine. The lab tests were performed with no knowledge whatsoever of the questionnaire responses. Based on previous studies (Jarvis et al., 1992; Cook et al., 1994; Dell'Orco et al., 1995; Bakoula et al., 1997), we analyzed the urinary cotinine levels in children with reference to the day of week on which the sample was collected.

Statistical Analysis

A bivariate analysis of variance (ANOVA) was used in order to identify which factors were associated with cotinine levels in children. In the case of quantitative variables, the analysis of simple linear regression and the coefficient of linear correlation of Pearson were used. Multiple regression analysis was used to evaluate the effect of each variable on CCR. The analysis included all the variables with statistical significance (P < 0.05), as well as those that gave nearly significant in the bivariate analysis. The parameters used were the coefficient of partial multiple correlation (*r*-partial) and the R^2 coefficient of determination, which corresponds to the proportion of variance of the dependent variable explained by the model. We took special care with colinearity between independent variables, so when a situation of clear colinearity was found, the independent variable most strongly associated with the variable of response was chosen.

The cotinine/creatinine ratio was log-retransformed to verify statistical hypotheses of variance homogeneity of the ANOVA and linear regression models. Geometric means were used in the tables to represent the data. All statistical analyses were carried out with the software package SPSS 9.01.

Results

The mean age of the children was 4.9 years (SD 0.9), with a higher proportion of boys (45.2%) than of girls (37.4%). The gender of 20 (17.4%) children was not specified on the returned questionnaire. A total of 59 (51.3%) of the mothers and 58 (50.4%) of the fathers were current smokers. Of the 115 children, 50 (43.5%) had two smoking parents. Moreover, 75 (65.2%) children were exposed to ETS in the household according to number of cigarettes/day smoked by all family members (at least 1–5 cigarettes/day).

A strong variability in the distribution of CCR levels is seen in Figure 1. The CCR levels ranged from 0 to 1014 ng/ mg with an arithmetic mean of 85.03 ng/mg. Considering a limit of detection of 10 ng cotinine/mg creatinine (see Chilmonczyk et al., 1993), 30 (26%) of the 115 children children had no cotinine (27 children had 0 ng/mg and three children had between 0.5 and 0.7 ng/mg), whereas cotinine was detected in the other 85 (74%).

Moreover, in 17 (13.8%) children whose parents smoked (one or both parents), no CCR was detected, and in 16 (14%) children urinary cotinine was detected despite the fact

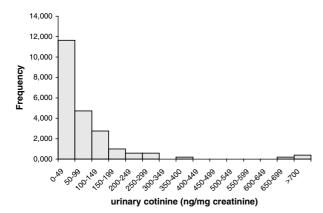


Figure 1. Urinary cotinine levels in children.

Table 1. Urinary	cotinine	levels	in	children	according	to	socio-
demographic varia	bles and c	day of	wee	k of sam	ple collected	1.	

Variable	N^{a}	Geometric mean	Geometric SD	DF	F	P-value
Age of children						
(years)						
4	27	23.1	7.9			
5	34	19.5	8.6			
6	39	32.2	7.4			
>6	11	29.0	3.5	3	0.41	0.746
Sex of children						
Male	52	27.9	7.9			
Female	43	26.0	6.7	1	0.03	0.859
Father's education						
Primary	33	37.3	6.2			
Secondary	15	28.5	6.6			
Technical	28	29.5	7.7			
University	26	12.9	8.9	3	0.85	0.471
Mother's education	ı					
Primary	37	30.4	6.9			
Secondary	18	36.0	8.0			
Technical	22	22.7	8.1			
University	34	18.2	7.3	3	0.55	0.649
Index of crowding						
<1	40	19.1	6.1			
= 1	47	21.3	6.6			
>1	18	56.3	9.3	2	3.45	0.036
Days of the week						
Tuesday	46	41.9	6.0	3	5.85	0.001
Wednesday	46	19.4	7.6			
Thursday	10	4.7	7.7			
Friday	10	31.5	7.4			

^aNumbers do not add up to 115 children and families due to missing values.

that the parents had affirmed they were nonsmokers or exsmokers.

Table 1 shows the sociodemographic factors associated with the urinary cotinine levels in children. There was a modest increase in CCR with age, and the levels were higher in males than in females. There was a tendency of decreasing CCR levels with increasing educational status of the parents, and although no statistically significant association could be established, this variable was considered in the linear multiple regression analysis. Children living in a house with a crowding index over 1 had significantly higher levels of cotinine than those who lived in homes with indexes of 1 or under. Furthermore, we found that urine samples collected on Tuesdays had significantly higher concentrations of cotinine than those taken on Wednesdays, Thursdays or Fridays.

The smoking status of the father was not associated with CCR, although there were signs of statistical significance (Table 2). There was a clear association between CCR levels and current smoking mothers: their offspring had higher levels of cotinine than the children whose mothers were nonsmokers. The CCR increased significantly with the

Table 2.	Urinary	cotinine	levels in	children	according	to	parents'	smoking	behavior	at	home.

Variable	N^{a}	Geometric mean	Geometric SD	DF	F	P-value
Father's smoking status						
Nonsmoker	24	12.1	6.5			
Ex-smoker	14	31.4	10.3			
Occasional	9	9.6	13.7			
Current smoker	58	34.1	6.7	3	2.00	0.119
Mother's smoking status						
Nonsmoker	49	14.4	7.4			
Ex-smoker						
Occasional	2	99.8	1.2			
Current smoker	59	39.3	6.7	2	4.31	0.015
Number of smoking parents						
None	30	9.2	6.2			
One	35	18.8	7.5			
Both	50	44.7	6.6	2	5.35	0.006
Father smoking in the living room in presence of children						
Yes	36	69.9	8.2			
No	21	11.7	3.8	1	15.28	< 0.001
Mother smoking in the living room in presence of children						
Yes	31	70.5	2.1	1	5.18	0.027
No	29	24.2	1.5			
Father smoking on patio, balcony						
Yes	32	63.8	3.0			
No	25	20.7	9.7	1	5.19	0.027
Mother smoking on patio, balcony						
Yes	40	41.4	6.7			
No	19	39.5	6.8	1	0.01	0.932
Father smoking at home (cigarettes/day)						
0	5	13.0	6.5			
1–5	43	29.3	6.4			
>5	10	126.6	3.0	2	6.43	0.003
Mother smoking at home (cigarettes/day)						
0	4	47.4	9.1			
1–5	41	32.3	6.4			
>5	14	94.0	5.0	2	1.58	0.216
Cigarettes/day smoked at home by all family						
0	30	8.4	8.5			
1–5	35	26.5	6.0			
6–10	20	58.3	4.4			
11–20	10	22.4	10.8			
>21	10	107.1	2.6	4	4.11	0.004
Parental perception of smokiness at home						
None at all	52	13.6	8.1			
A little	49	36.6	6.1			
Quite a bit/a lot	6	161.2	2.8	2	4.38	0.015

^aNumbers do not add up to 115 children and families due to missing values.

number of smoking parents (Pearson's correlation coefficient r = 0.308; P = 0.001).

The practices of the parents at home also affected the cotinine levels. There were statistically significant increases in the mean levels of urinary cotinine in children whose fathers affirmed smoking in the living room in the presence of child, and the number of cigarettes/day that the father smoked inside the house. However, smoking in another part of the house (patio or balcony) was also associated with higher cotinine concentrations. Among mothers, smoking in the

living room showed an association with CCR. The number of cigarettes/day that the family (parents and others) smoked at home significantly influenced the mean levels of urinary cotinine in children. Moreover, there was a noteworthy association between the smoky environment perceived by all parents (smokers, nonsmokers) and the CCR (Table 2).

Table 3 presents the results of multiple linear regression analysis. The final regression model (model II) included four variables: father's education, mother's smoking status, parental perception of smokiness at home, and day of week

Variable	Model I		Model II		
	r-partial	<i>P</i> -value	r-partial	P-value	
Father's education	-0.121	0.269	-0.208	0.050	
Mother's education	-0.041	0.707			
Index of crowding	0.118	0.284			
Day of week of sample collected	0.221	0.042	0.263	0.012	
Mother's smoking status	0.069	0.528	0.159	0.134	
Number of smoking parents	0.049	0.653			
Parental perception of smokiness at home	0.284	0.008	0.324	0.002	

Table 3. Multiple linear regression analysis of factors associated with urinary cotinine in children.

r-partial, coefficient of partial correlation; P, significance value; R^2 , coefficient of determination; Model I: $R^2 = 0.299$; Model II: $R^2 = 0.264$.

of sample. The educational level of the father was a significant influence (P < 0.05): the higher his level of studies, the lower the cotinine levels in the child. The children whose mothers admitted smoking daily had higher levels of cotinine than the children whose mothers did not. The atmosphere of smokiness in the home had a significant influence on the level of cotinine in the children (P < 0.002), and increased progressively with the degree of smokiness described by the parents (from "none at all" to "a lot"). Tuesday as the sampling day also exerted an independent influence (P=0.012) on cotinine. The four variables present in this model explained 26.4% of the variance in the cotinine levels of children. In the group where the perception of smokiness in the home was described as "quite a bit/a lot", there were only six children, which might suggest that the elimination of these data would drastically change the model. When these data were eliminated, however, the determination coefficient was 0.243, representing only a minor decrease in the explained variance. Likewise, the partial correlation coefficient of the parental perception of smokiness showed little decrease when these six cases were eliminated (r-partial coefficient = 0.274). For this reason, we maintained the four cited variables in the final model.

Discussion

Questionnaires about parents' smoking behavior and studies of urinary cotinine levels are widely used to estimate the passive smoking of children (see Introduction). In this study, we used both methods to assess exposure to ETS in 115 children aged 3–6 years. Overall, 65.2% of these children were exposed to tobacco smoke on the basis of the number of cigarettes/day reportedly smoked by all family members inside the house (at least 1–5 cigarettes/day). When exposure to ETS was evaluated by urinary cotinine levels, the proportion of children exposed was 74%. The fact that no cotinine was detected in the urine of 17 (13.7%) children of smoking parents (one or both) could be because these parents smoked mainly outside the home. A similar finding has been noted by Winkelstein et al. (1997) and Seifert et al. (2002). In addition, cotinine was present in the urine of 16 (14%)children whose parents had stated they were nonsmokers or ex-smokers. This might be attributed to exposure to other smokers in the family (in 93% of the homes there was a smoking family member habitually present, regardless of whether the parents smoked) as other authors have found (Cook et al., 1994; Dell'Orco et al., 1995; Irvine et al., 1997; Preston et al., 1997; Jordaan et al., 1999; Ownby et al., 2000); or exposure to nonhousehold smokers (Cook et al., 1994; Dell'Orco et al., 1995; Jordaan et al., 1999). Nonetheless, we might also suspect that some parents were not completely forthright in describing their smoking habits, an argument underlined by previous authors (Peterson et al., 1997; Seifert et al., 2002).

As expected, the urinary cotinine levels increased significantly with the number of smoking parents — up to five times higher — in the children who had two smoking parents (geometric mean = 44.7 ng/mg creatinine) than among those whose parents did not smoke (geometric mean = 9.2 ng/mgcreatinine). This finding is in accordance with data of other authors (Cook et al., 1994; Irvine et al., 1997; Seifert et al., 2002).

The results of our study confirm the influence of maternal smoking in the cotinine levels in children, a well-documented finding (Cook et al., 1994; Dell'Orco et al., 1995; Irvine et al., 1997; Preston et al., 1997; Jordaan et al., 1999; Oddoze et al., 1999), but paternal smoking also played a important role in the exposure of the child, as some other studies have shown (Dell'Orco et al., 1995; Jordaan et al., 1999). Indeed, the specific practices at home of both fathers and mothers, such as smoking in the living room in the presence of child, were responsible for increases in cotinine levels. This finding is in agreement with the study of Irvine et al. (1997), and with the more recent study of Seifert et al. (2002). Curiously enough, when the father reported smoking on patio or balcony, the urinary cotinine of the child was significantly higher than in children whose parents did not report this practice. This

result, also observed by Bahçeciler et al. (1999), may indicate that some fathers are not telling the whole truth, but acknowledge that it is better to avoid exposing their children to smoke.

The total number of cigarettes per day smoked by all family members (parents and other usual residents) inside the house was associated with cotinine, in accordance with previous studies (Preston et al., 1997; Winkelstein et al., 1997; Manino et al., 2001; Callais et al., 2003).

Our study confirms the findings from several other studies (Jarvis et al., 1992; Cook et al., 1994; Dell'Orco et al., 1995; Bakoula et al., 1997; Irvine et al., 1997; Jordaan et al., 1999; Manino et al., 2001) in the socioeconomic factors are associated with cotinine levels. The children whose fathers were better educated and children living in a home with one person or less per room (crowding index ≤ 1) had significantly lower levels of cotinine.

The family atmosphere played a very important role in urinary cotinine of children, to such a point that on Tuesday, two days after the weekend (when family contact is greatest), elevated levels of cotinine were still present in the children's urine. This stands as objective evidence of exposure in the household. Cotinine is known to have a half-life of 16–19 h, and it can be detected in organic fluids even 2 or 3 days after exposure (Benowitz, 1996). A similar finding is documented by other researchers (Jarvis et al., 1992; Cook et al., 1994; Dell'Orco et al., 1995; Bakoula et al., 1997): they found significantly higher cotinine levels in the urine samples collected on Mondays. Jordaan et al. (1999), who took samples only on Wednesdays and Thursdays, found no association between cotinine levels and day of the week.

The perception of parents (smokers and nonsmokers) of smokiness in the home was, interestingly enough, the variable exhibiting the greatest influence on the levels of cotinine in children. This result shows the discriminating potential of the question posed: "How would you classify the atmosphere of tobacco smoke usually present in your home?" Although what is being measured is a perception (subjective sphere of the individual), the assessment is made in frank quantitative terms (none at all, a little, quite a bit, a lot), which may prove to be less provocative of parental denial or justification of their smoking behavior. We could find no studies with similar examples of how parental perceptions can be determinant of exposure to ETS except Dell'Orco et al. (1995), who studied the perception of a group of Italian adolescents about smokiness at home over the three days previous, and found an association between this perception and the levels of cotinine in urine of the adolescents. Our results and those of Dell'Orco et al., (1995) demonstrate that the perception about smokiness at home can play an important role as an indicator of children's exposure to household tobacco smoke.

We conclude that it is necessary to analyze those factors related with cotinine levels in children when designing

educational programs to reduce passive smoking among children. The results of our study confirm the association between cotinine in children and the mothers' smoking status (Jordaan et al., 1999), the fathers' educational level (Dell'Orco et al., 1995; Manino et al., 2001), and the day of the week of sampling (Jarvis et al., 1992; Dell'Orco, 1995; Bakoula et al., 1997). Furthermore, we arrive at a new factor, the perception of parents (smokers and nonsmokers) about the smokiness in the home, which also influences the cotinine levels and may prove a useful indicator of children's exposure to household tobacco smoke.

References

- Aligne C.A., and Stoddard J.J. Tobacco and children. An economic evaluation of the medical effects of parental smoking. *Arch Pediatr Adolesc Med* 1997: 151: 648–653.
- Bahçeciler N.N., Barlan I.B., Nuhoglu Y., and Basaran M.M. Parental smoking behavior and the urinary cotinine levels of asthmatic children. J Asthma 1999: 36: 171–175.
- Bakoula C.G., Kafritsa Y.J., Kavadias G.D., Haley N.J., and Matsaniotis N.S. Factors modifying exposure to environmental tobacco smoke in children (Athens, Greece). *Cancer Cause Control* 1997: 8: 73–76.
- Benowitz N.L. Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev* 1996: 18: 188–204.
- Callais F., Momas I., Roche D., Gauvin S., Reungoat P., and Zmirou D. Questionnaire or objective assessment for studying exposure to tobacco smoke among asthmatic and healthy children: the French VESTA study. *Prev Med* 2003: 36: 108–113.
- Chilmonczyk B.A., Salmun L.M., Megathlin K.N., Neveux L.M., Palomaki G.E., and Knight G.J., et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. N Engl J Med 1993: 328: 1665–1669.
- Cook G.C., Wincup P.H., Jarvis M.J., Strachan D.P., Papacosta O., and Bryant A. Passive exposure to tobacco smoke in children aged 5–7 years: individual, family, and community factors. *Br Med J* 1994: 308: 384–389.
- Dell'Orco V., Forastiere F., Agabiti N., Corbo G.M., Pistelli R., and Pacifici R., et al. Household and community determinants of exposure to involuntary smoking: a study of urinary cotinine in children and adolescents. *Am J Epidemiol* 1995: 142: 419–427.
- Henderson F.W., Reid H.F., Morris R., Wang O.L., Hu P.C., and Helms R.W. Home air nicotine levels and urinary cotinine excretion in preschool children. *Am Rev Respir Dis* 1989: 140: 197–201.
- Irvine L., Crombie I.K., Clark R.A., Slane P.W., Goodman K.E., and Feyerabend C., et al. What determines level of passive smoking in children with asthma? *Thorax* 1997: 52: 766–769.
- Hovell M.F., Zakarian J.M., Wahlgren D.R., Matt G.E., and Emmons K.M. Reported measures of environmental tobacco smoke exposure: trials and tribulations. *Tob Control* 2000: 9(Suppl 3): III22–28.
- Jarvis M.J., Strachan D.P., and Feyerabend C. Determinants of passive smoking in children in Edinburgh, Scotland. Am J Public Health 1992: 82: 1225–1229.
- Jordaan E.R., Ehrlich R.I., and Potter P. Environmental tobacco smoke exposure in children: household and community determinants. *Arch Environ Health* 1999: 54: 319–327.
- Manino D.M., Caraballo R., and Benowitz N. Repace J. Predictors of cotinine levels in US children: data from the Third National Health and Nutrition Examination Survey. *Chest* 2001: 120: 718–724.

- Oddoze C., Dubus J.C., Badier M., Thirion X., Pauli A.M., and Pastor J., et al. Urinary cotinine and exposure to parental smoking in a population of children with asthma. *Clin Chem* 1999: 45: 505–509.
- Ownby D.R., Johnson C.C., and Peterson E.L. Passive cigarette smoke exposure of infants: importance of nonparental sources. *Arch Pediatr Adolesc Med* 2000: 154: 1237–1241.
- Peterson E.L., Johnson C.C., and Ownby D.R. Use of urinary cotinine and questionnaires in the evaluation of infant exposure to tobacco smoke in epidemiologic studies. *J Clin Epidemiol* 1997: 50: 917–923.
- Preston A.M., Ramos L.J., Calderon C., and Sahai H. Exposure of Puerto Rican children to environmental tobacco smoke. *Prev Med* 1997: 26: 1–7.
- Seifert J.A., Ross C.A., and Norris J.M. Validation of a five-question survey to assess a child's exposure to environmental tobacco smoke. *Ann Epidemiol* 2002: 12: 273–277.

- U.S. Department of Health and Human Services, Public Health Service, Center for Disease Control. Cigarette smoking among adults — United States, 1993. *MMWR* 1994: 43: 926–948.
- US Environmental Protection Agency. *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*. Environmental Protection Agency, Washington, DC, 1992.
- Winkelstein M.L., Tarzian A., and Wood R.A. Parental smoking behavior and passive smoke exposure in children with asthma. *Ann Allergy Asthma Immunol* 1997: 78: 419–423.
- World Health Organization Division of Noncommunicable Diseases. Tobacco Free Initiative. International consultation on environmental tobacco smoke (ETS) and child health. *Consultation Report*. WHO, Geneva, 1999.